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Responses of Exercising Subjects to Acute "Passive" Cigarette Smoke Exposure

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Responses to 2 hr of "passive" cigarette smoke exposure have been tested in 23 healthy young men and women who were performing intermittent bicycle ergometer work sufficient to increase respiratory minute volumes by a factor of 2.5. A simple crossover design compared data with reactions to sham exposures of similar duration. Cigarettes were smoked by a standard machine, chamber carbon monoxide concentrations were 20 (moderate dose) or 31 ppm (heavy exposure). Symptoms were much as in moderate exposures without exercise. The main complaints were of odor and eye irritation. Cough, nasal discharge or stuffiness, and throat irritation were also reported, but wheezing, shortness of breath, and tightness in the chest were both uncommon and unsupported by objective evidence of bronchospasm. A small increase of tidal volume and respiratory minute volume seemed due to anxiety rather than airway irritation. Static lung volumes were unchanged, but there were small (3–4%) decreases of FVC, $FEV_{1.0}$, $\dot{V}_{max\ 50\%}$, and $\dot{V}_{max\ 25\%}$. The changes of dynamic lung volumes were of the order anticipated from the "cigarette equivalent" encountered by the passive smoker (<½ cigarette in 2 hr).

INTRODUCTION

Appreciable atmospheric concentrations of particulate matter, irritant gases, and vapors can develop due to the accumulation of tobacco smoke in poorly ventilated buildings (for references, see Sebben *et al.*, 1977; Pimm *et al.*, 1978; Shephard *et al.*, 1978a). The "passive" exposures to cigarette smoke are subjectively unpleasant for most nonsmokers plus some continuing and former smokers (Johansson and Rouge, 1965; Anderson and Dalhamn, 1973; Weber-Tschopp *et al.*, 1976; Shephard and LaBarre, 1978), leading to irritation of the eyes and nose (Shephard *et al.*, 1978b,c) and instability of the tear film (Basu *et al.*, 1978). Some authors have also described cardiac and respiratory symptoms (Aronow, 1978; Surgeon General, 1972; Pimm *et al.*, 1977, 1978), an increased incidence of respiratory infections in the children of smoking parents (Norman-Taylor and Dickinson, 1972; Cameron and Robertson, 1973; Colley, 1974; Harlap and Davies, 1974), and an increased risk of lung cancer (British Medical Journal, 1978). However, other investigators have stressed that the increase of ambient CO concentration in a room contaminated by cigarette smoke is quite small (Fischer *et al.*, 1978), particularly if allowance is made for the effect of interfering vapors such as ethanol (First and Hinds, 1976) upon the usual CO-measuring instrument (the "Ecolyser"). Further, one recent study has failed to confirm the supposed effect of parental smoking upon the respiratory health of children (Schilling *et al.*, 1977).

Experimental exposures to moderate concentrations of cigarette smoke have demonstrated only small changes of pulmonary function, in some instances statistically significant, but of doubtful biological importance (Pimm *et al.*, 1978). Since

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changes of respiratory mechanics are an early acute response to smoking in both smokers and nonsmokers (Nadel and Comroe, 1961; Clarke *et al.*, 1970; Da Silva and Hamosh, 1973; Hamosh and Da Silva, 1977), it was decided to examine the pulmonary reactions of the "passive" smoker under more adverse conditions than those previously evaluated. The respiratory minute volume during exposure was increased by intermittent moderate exercise, and in a final series of experiments the number of cigarettes burnt in the experimental chamber was also increased.

METHODS

Subjects and experimental plan. The subjects were 23 healthy young adult volunteers, drawn from the University of Toronto Community. Physical characteristics are summarized in Table 1. All were life-long nonsmokers (cigarette consumption nil for the past year, no history of smoking > one cigarette per day). None had any history of allergic disease.

A preliminary visit to the laboratory permitted clinical examination and familiarization of the subjects with the required test procedures. At comparable times on 2 subsequent days, subjects spent 2 hr in an exposure chamber, alternating 15-min periods of exercise sufficient to increase respiratory minute volume by a factor of 2.5 with sitting at rest. The chamber was filled with either ambient air (sham exposure) or cigarette smoke (experimental exposure), concentrations being as in previous experiments (Pimm *et al.*, 1978) for the first 13 subjects and augmented by some 50% for the second group of 10 volunteers.

The protocol followed for the 2 exposure days is summarized in Table 2.

Exposure conditions. Details of the exposure chamber are given in a previous report (Pimm *et al.*, 1978). In brief, a standard cigarette smoking machine (Weder and Hofmann, 1967) was operated in a sparsely furnished 14.6 m^3 chamber. A popular brand of 85-mm filter-type cigarette (tar and nicotine content 19 and 1 mg, respectively) was smoked by brisk (2-sec) controlled 35-ml draws to a butt length of 23 mm. For the first 13 subjects, four cigarettes were burnt initially, followed by a further cigarette at half-hour intervals. For the second 10 subjects the initial combustion was increased to six cigarettes, again followed by the burning of one further cigarette at half-hour intervals.

The first pattern of combustion yielded a carbon monoxide concentration of 20.0 ± 1.6 ppm for the men and 20.1 ± 2.4 ppm for the women, with particulate levels declining slowly from 4 to 2 mg/m^3. With the six cigarettes, air contami-

TABLE 1
PHYSICAL CHARACTERISTICS OF SUBJECTS (MEAN ± SD OR DATA)

	Age (years)	Height (cm)	Weight (k
Males			
(4 → 3 cigarettes, n = 6)	22.7 ± 3.2	177.0 ± 9.9	67.9 ± 12
(6 → 3 cigarettes, n = 5)	23.4 ± 4.0	172.4 ± 9.0	73.6 ± 22
Females			
(4 → 3 cigarettes, n = 7)	24.1 ± 4.0	160.0 ± 10.6	51.1 ± ?
(6 → 3 cigarettes, n = 5)	27.4 ± 5.1	160.0 ± 12.4	50.2 ± ?

TABLE 2
PROTOCOL FOR 2-hr CHAMBER EXPOSURE TO AIR OR CIGARETTE SMOKE

Prechamber tests	Lung volumes by helium dilution (reduced VC, RV, FRC, ERV, TLC)
	Carboxyhemoglobin by rebreathing technique
	Resting ventilation ($\dot{V}_E$, f_R, V_T)
	Flow-volume curves (FVC, $FEV_{1.0}$, $\dot{V}_{max\ 25\%}$, $\dot{V}_{max\ 50\%}$)
	Resting electrocardiogram
Chamber tests	Pedal bicycle ergometer at load to increase $\dot{V}_E$ to 2.5 times resting level 11–26 min, 41–56 min, 71–86 min, 101–116 min
	Flow-volume curves at 0, 5, 10, 30, 60, 90, 120 min
	Resting ventilation at 30, 40, 60, 90, 100, 120 min
	Exercise ventilation at 54, 84, 114 min
	Electrocardiogram every 10 min, 20–120 min
Preexposure tests	Lung volumes by helium dilution
	Carboxyhemoglobin
	Symptom questionnaire

tion was some 50% greater, CO levels averaging 31.1 ± 4.3 ppm for the men, and 31.4 ± 4.0 ppm for the women.

Lung volumes. Functional residual capacity (FRC), expiratory reserve (ERV), residual volume (RV), and total lung capacity (TLC) were measured by means of the 7-min helium rebreathing technique (Collins respirometer/catharometer system).

Respiratory minute volume ($\dot{V}_E$), breathing frequency (f_R), and maximum expiratory flow-volume curves were obtained using a heated Fleisch (No. 3) pneumotachograph and integrator, volume and flow signals being displayed on a Tectronix storage oscilloscope. At each test period, the subject was seated, and performed three forced vital capacity (FVC) maneuvers, the curve with the largest FVC being used for analysis. In addition to FVC, measurements were taken of 1-sec forced expiratory volume ($FEV_{1.0}$) and the maximum flow at 25% ($\dot{V}_{max\ 25\%}$) and 50% ($\dot{V}_{max\ 50\%}$) of the vital capacity.

Exercise. Subjects performed standard intermittent exercise on a Von Döbeln bicycle ergometer. In order to avoid any complication from exercise-induced bronchospasm, the 15-min periods of exercise were displaced as far as possible from respiratory function measurements. Work loads were set to yield approximately a 2.5-fold increase of respiratory minute volume. Taking account also of the intervening 15-min rest periods, the average respiratory minute volume over the experiment was approximately 1.75 times the normal resting level. Heart rates during exposure were recorded by electrocardiogram (CM_5 lead, Shephard, 1977).

Symptoms. At the end of each exposure, subjects were asked to respond in a yes/no fashion to the presence of several potential symptoms (Table 3). In the event that a symptom was reported, they were then asked to rate its severity (trace, minimal, moderate, severe, or very severe).

Statistical analysis. All data were expressed as percentages of the preexposure reading for the corresponding day. Differences between test and control days were assessed using standard *t* tests.

TABLE 3

SUBJECTIVE REACTIONS TO CIGARETTE SMOKE—2-hr EXPOSURES TO 4 ± 3 AND 6 ± 3 CIGARETTES WITH INTERMITTENT EXERCISE

Symptom	4 ± 3 Cigarettes (n = 12)[a]						6 ± 3 Cigarettes (n = 10)					
	Nil	Trace	Minimal	Moderate	Severe	Very severe	Nil	Trace	Minimal	Moderate	Severe	Very severe
Use of medication	12	0	0	0	0	0	10	0	0	0	0	0
Odor	0	0	1	5	2	2	1	0	0	1	3	5
Nausea	12	0	0	0	0	0	10	1	1	0	0	0
Cough	5	2	4	1	0	0	6	1	1	2	0	0
Sputum	11	1	0	0	0	0	0	0	0	0	0	0
Soreness												
Substernal	12	0	0	0	0	0	9	0	1	0	0	0
Muscular	12	0	0	0	0	0	10	0	0	0	0	0
Other	12	0	0	0	0	0	10	0	0	0	0	0
Throat	7	3[a]	2	0	0	0	7	1[a]	1	1	0	0
Shortness of breath	10	1	0	1	0	0	8	1	0	1	0	0
Nasal discharge or stuffiness	8	2	1	1	0	0	4	1	1	2	1	0
Wheezing	12	0	0	0	0	0	9	1	0	0	0	0
Tightness in chest	12	0	0	0	0	0	9	0	1	0	0	0
Dizziness	9	1	2	0	0	0	6	2	1	1	0	0
Fatigue	10	1	0	1	0	0	8	0[a]	2[a]	0	0	0
Eye irritation	1	1	1	1	6	2	1	0	1	0	4	4
Headache	9	0	2	1	0	0	7	0	1	1	1	0
Other	12	0	0	0	0	0	0	0	0	0	0	0
Total	165	12	13	11	8	4	115	8	11	9	9	9

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RESULTS

Symptoms

At both levels of exposure, the main complaints were of odor and eye irritation (Table 3). Even at the highest dose, only one subject (D.K.) complained of wheezing and tightness in the chest; he did not show any unusual impairment of flow/volume curves, although at the end of the 2-hr exposure (120, 114 min) his resting and exercise $\dot{V}_E$ were 143% and 154%, respectively, of the corresponding control values.

Awarding points of 0 to 5 according to the severity of the symptoms reported, 12 subjects (1 subject failed to report the symptoms encountered), accumulated a total of 123 points (10.3 points/subject) at the lower level of exposure, while the 10 subjects exposed to the higher smoke concentration had a marginally greater score of 138 points (13.8 points/subject).

Cardiorespiratory Performance

The respiratory minute volume of most subjects was quite high before they entered the exposure chamber (Table 4). Nevertheless, values were further increased by the cigarette smoke but not by the sham exposure. In the 4 + 3 cigarette experiments, the increase of ventilation relative to sham averaged 9.6% during exercise (NS), and was 21.4% during the intervening rest periods ($P > 0.025$). In calculating the significance of these trends, data for each individual have been averaged over time, and the difference between these averaged responses for experimental and sham exposures has been calculated by standard two-tailed t statistics. In the 6 − 3 cigarette experiments, the effect was no greater (11.1% during exercise, NS; 12.5% during recovery, NS).

Any increase of respiratory minute volume was almost entirely attributable to an increase of tidal volume. The initial, preexposure respiratory rate was greater than normal. Comparing sham and experimental exposures, cigarette smoke was associated with an insignificant decrease of f_R during exercise (−1.8 and −6.4% at the two exposure levels), while during the recovery intervals there was an insignificant increase (3.4 and 3.7% at the two levels).

The heart rate was higher before the experimental than before the sham exposures (Table 5; for women in 4 + 3 cigarette experiment, $P < 0.01$, for men in 6 + 3 cigarette experiment $P < 0.025$). However, while actually exposed to the cigarette smoke both the increment of heart rate and the absolute heart rate were less than in the corresponding sham exposure.

Static Lung Volumes

In both the 4 + 3 and the 6 + 3 cigarette experiments (Table 6), the preexposure static lung volumes did not differ between sham and experimental days. There was a slight suggestion that cigarette smoke led to a decrease of expiratory reserve volume and functional residual capacity as indicated by helium mixing, but this trend was statistically insignificant.

Dynamic Lung Volumes

The preexposure dynamic lung volumes (Table 7) did not differ between sham and experimental days. In the 4 − 3 cigarette experiments, all results obtained during exposure were somewhat depressed on experimental days relative to sham

TABLE 4

RESPIRATORY MINUTE VOLUME ($\dot{V}_E$) AND BREATHING FREQUENCY (f_R)[a]

Variable			Preexposure period	Exposure period: Rest[b] (%)	Exercise[b] (%)
			4 + 3 Cigarette experiment (n = 11)[c]		
$\dot{V}_E$	(S)	Men	16.1 ± 4.7 l · min^{-1}	105.7	241.7
		Women	8.7 ± 2.0 l · min^{-1}		
	(E)	Men	98 ± 29%	128.3[d]	264.0
		Women	105 ± 21%		
f_R	(S)	Men	25 ± 8 breaths · min^{-1}	103.3	128.3
		Women	17 ± 4 breaths · min^{-1}		
	(E)	Men	102 ± 33%	106.8	126.0
		Women	94 ± 30%		
			6 + 3 Cigarette experiment (n = 7)[c]		
$\dot{V}_E$	(S)	Men	14.8 ± 4.6 l · min^{-1}	95.4	214.3
		Women	11.3 ± 2.9 l · min^{-1}		
	(E)	Men	92 ± 32%	107.3	238.3
		Women	77 ± 26%		
f_R	(S)	Men	14.7 ± 3.1 breaths · min^{-1}	96.2	162.3
		Women	20.5 ± 5.3 breaths · min^{-1}		
	(E)	Men	92 ± 30%	99.8	152.0
		Women	124 ± 52%		

[a] Mean ± SD of data for last preexposure period in sham exposure S (absolute values, l · min^{-1} BTPS and breaths · min^{-1}), (b) preexposure in experimental exposure E (percentage of sham preexposure periods), and (c) sham and experimental exposures (percentage of corresponding preexposure periods).

[b] Resting data for 30, 40, 60, 90, 100, 120 min of exposure averaged. Exercise data for 34, 84, 114 min of exposure averaged. Individual data with SD available on request.

[c] Complete data not available for remaining subjects.

[d] Increase over sham exposure $P < 0.025$.

days, with a 5.6% decrease of FVC ($0.2 > P > 0.1$), a 3.3% decrease of $FEV_{1.0}$ ($P < 0.05$), a 4.2% decrease of $\dot{V}_{max\ 50\%}$ ($0.1 > P > 0.05$), and a 4.8% decrease of $\dot{V}_{max\ 25\%}$ ($0.2 > P > 0.1$). There was little evidence of adaptation to the cigarette smoke over the 2-hr exposure—indeed, the final results showed a slightly greater functional loss than those observed in the first few minutes of the experiment.

In the 6 + 3 cigarette experiments, the FVC again tended to be depressed (average change 3.2%, NS), as was the $\dot{V}_{max\ 25\%}$ (average change 8.0%). Changes in the $\dot{V}_{max\ 50\%}$ and $FEV_{1.0}$ were variable and statistically insignific

DISCUSSION

Severity of Dosage

The levels of passive cigarette smoke exposure selected for this investigation were not the highest reported values for contaminated rooms and vehicles: some authors have encountered CO concentrations of 60–156 ppm (Wahl, 1 Harmsen and Effenberger, 1957; Srch, 1967; Hoegg, 1972; Hanke, 1

TABLE 5
HEART RATE DATA[a]

Type of exposure		Preexposure period	Exposure period: Rest[b] (%)	Exposure period: Exercise[b] (%)
		4 × 3 Cigarette experiment (n = 10)[c]		
(S)	Men	72 ± 8 beats · min^{-1}	121.3	177.0
	Women	77 ± 15 beats · min^{-1}		
(E)	Men	106 ± 8%[d]	100.9	137.8
	Women	115 ± 7%[d]		
		6 × 3 Cigarette experiment (n = 9)[c]		
(S)	Men	64 ± 8 beats · min^{-1}	116.9	170.3
	Women	74 ± 9 beats · min^{-1}		
(E)	Men	124 ± 18%[e]	112.3	156.3
	Women	109 ± 20%[e]		

[a] Mean ± SD for (a) preexposure period in sham exposure S (absolute values, beats · min^{-1}), (b) preexposure period in experimental exposure E (percentage of sham preexposure period), and (c) sham and experimental exposures (percentage of corresponding preexposure period).

[b] Resting data for 30, 40, 60, 70, 90, 100, 120 min of exposure averaged. Exercise data for 20, 50, 80, 110 min of exposure averaged. Individual data with SD available on request.

[c] Complete data not available for remaining subjects.

[d] Increase over sham exposure, $P < 0.01$.

[e] Increase over sham exposure, $P < 0.05$.

Nevertheless, they seem realistic in the context of air quality criteria, representing the greatest likely hazard that would be encountered by a person undertaking moderate physical work in a smoke-contaminated and poorly ventilated room such as a tavern (Sebben *et al.*, 1977).

Symptoms Reported

Complaints arise in aircraft, trains, and buses when cigarette-induced increments of carbon monoxide concentration reach about a fifth of the values used in the present experiments (Sebben *et al.*, 1977; Shephard and LaBarre, 1978; Shephard *et al.*, 1978a). Furthermore, the complainants are usually sitting, rather than undertaking intermittent exercise. It is thus hardly surprising that our subjects had some complaints. What is more interesting is that as in more moderate exposures, comments often remained confined to odor and eye irritation. About a half of the subjects noticed some coughing, and there were also reports of nasal discharge or stuffiness and throat irritation. However, only one subject had responses indicative of bronchospasm (wheezing and shortness of breath). The rarity of a subjectively detectable increase of airway resistance may be explained on the basis that subjects undertaking light work fail to detect less than a fourfold increase of airway resistance (McKerrow *et al.*, 1958).

An increase in the initial combustion of cigarettes did not lead to any great increase in the number or severity of reported symptoms. Possibly, there was a saturation or adaptation of the receptors concerned. Certainly, conjunctival irritation was most marked on first entering the exposure chamber, and became less

TABLE 6
STATIC LUNG VOLUMES[a]

Measurement		Relaxed vital capacity	Expiratory reserve volume	Functional residual capacity	Residual volume	Total lung capacity
	4 + 3 Cigarette experiment (n = 7, 10)[b]					
(S)	Pre Exp men	5.26 ± 1.13	1.86 ± 0.65	3.73 ± 1.02	1.87 ± 0.72	7.13 ± 1.17
	women	3.47 ± 0.58	1.23 ± 0.15	2.37 ± 0.68	1.14 ± 0.53	4.74 ± 0.99
	Exp men + women	102.7 ± 6.9%	107.4 ± 11.6%	101.0 ± 14.3%	98.7 ± 27.9%	99.9 ± 10.1%
(E)	Pre Exp men	100 ± 1%	107 ± 8%	96 ± 17%	88 ± 32%	96 ± 11%
	women	101 ± 5%	106 ± 10%	94 ± 7%	96 ± 9%	97 ± 5%
	Exp men + women	101.2 ± 3.9%	95.6 ± 6.9%	96.7 ± 12.7%	102.2 ± 32.9%	99.9 ± 6.9%
	6 + 3 Cigarette experiment (n = 7, 6)[b]					
(S)	Pre Exp men	5.74 ± 0.56	1.71 ± 0.41	2.88 ± 0.64	1.17 ± 0.24	6.90 ± 0.61
	women	3.37 ± 0.15	1.16 ± 0.06	2.29 ± 0.31	1.13 ± 0.36	4.44 ± 0.08
	Exp men + women	102.9 ± 4.8%	100.9 ± 13.6%	104.7 ± 14.9%	109.3 ± 20.0%	104.1 ± 3.3%
(E)	Pre Exp men	100 ± 1%	84 ± 17%	96 ± 4%	111 ± 20%	101 ± 5%
	women	106 ± 2%	118 ± 25%	106 ± 13%	96 ± 6%	103 ± 8%
	Exp men + women	101.2 ± 2.3%	95.7 ± 16.0%	98.2 ± 8.1%	100.5 ± 15.6%	103.0 ± 2.0%

[a] Mean ± SD of data for (a) preexposure period in sham exposure (S) absolute values, L (BTPS), (b) preexposure period in experimental exposure E (percentage of sham preexposure period), and (c) sham and experimental exposures (percentage of corresponding preexposure period).
[b] Complete data not available for remaining subjects.

as the experiment continued. The corneal pain receptors normally show a slow rate of adaptation, but it may be that the smoke became less irritant because it stimulated an increased lachrymal secretion (Shephard *et al.*, 1978c).

There is also a possibility that some or all of the other symptoms were "suggested" by the odor of cigarette smoke. In particular, it is surprising that the subject reporting wheezing and shortness of breath during exposure showed no significant decrement of objective measures of lung function. While this person may have had an unusual sensitivity to cigarette smoke, his symptoms could also have developed through a process of self-suggestion. There is thus scope for a definitive experiment relating objective measures of hypnotic susceptibility to the reported symptoms and physiological responses of the passive smoker.

Hyperventilation

At first inspection, the increase of respiratory minute volume during cigarette smoke exposure might seem to be objective evidence of airway irritation by the smoke particles. However, further examination of the data shows that this is an unlikely explanation, since the increment of $\dot{V}_E$ was attributable entirely to an increase of V_T. Stimulation of tracheal irritant receptors should curtail inspiratory drive, with a decrease of tidal volume and a compensatory increase of respiratory rate. We have described this pattern of response during inhalation of an irritant gas such as ozone (Folinsbee *et al.*, 1975). The increase of tidal volume could conceivably be attributed to a greater peripheral drive (for example, an action of absorbed nicotine upon the carotid chemoreceptors). However, the preexposure hyperventilation, the decreasing discrepancy between sham and experimental days as the exposure continued and the absence of a dose/response relationship all support an alternative hypothesis (a central facilitation of inspiratory drive by anxiety). A similar explanation would cover the slight tachycardia previously described in female subjects during exposure (Pimm *et al.*, 1978), and seen here before subjects entered the exposure chamber.

Static and Dynamic Lung Volumes

Static lung volumes show no consistent reaction to cigarette smoke exposure. However, the 4 + 3 cigarette experiments suggest a small decrement of dynamic lung volumes consistent with a small and practically unimportant decrease of airway conductance, while the 6 + 3 cigarette exposures induce statistically insignificant trends in the same direction.

Since dynamic airflow measurements depend upon the cooperation of the individual, it could be argued that there was some voluntary limitation of forced expiratory efforts in the smoke-filled room. Nevertheless, there are several pointers to a true pharmacological response:

(a) The male subjects in our previous resting exposures also showed a small decrease of $\dot{V}_{max\ 50\%}$ and $\dot{V}_{max\ 25\%}$ (Pimm *et al.*, 1978).

(b) The variance of the dynamic airflow measurements was comparable in experimental and sham exposures.

(c) The extent of flow impairment was consistent with previous observations made during active smoking.

Although Nadel and Comroe (1961) reported a 31% decrease of airway conductance in response to the smoking of a single cigarette, most authors have found

TABLE 7

CHANGES IN DYNAMIC LUNG VOLUMES DURING SHAM (S) AND EXPERIMENTAL (E) EXPOSURES[a]

			Preexposure	5 min (%)	120 min (%)	Average of seven observations[b]
			4 + 3 Cigarette experiment (n = 12)[c]			
FVC	(S)	Men Women	5.55 ± 1.69 liter 3.29 ± 0.57 liter	98.9 ± 6.4	99.7 ± 9.3	99.0
	(E)	Men Women	97 ± 11% 108 ± 7%	94.1 ± 7.5	92.9 ± 4.1	93.5
$FEV_{1.0}$	(S)	Men Women	4.17 ± 0.44 liter 2.94 ± 0.38 liter	101.7 ± 1.8	101.3 ± 7.0	101.7
	(E)	Men Women	93 ± 10% 100 ± 8%	95.7 ± 12.2	101.1 ± 9.1	98.1[d]
$\dot{V}_{max\ 50\%}$	(S)	Men Women	3.69 ± 0.64 liter · sec^{-1} 3.66 ± 0.92 liter · sec^{-1}	110.6 ± 19.7	110.2 ± 19.6	109.0
	(E)	Men Women	109 ± 10% 95 ± 15%	104.0 ± 9.4	102.7 ± 15.0	104.4[d]
$\dot{V}_{max\ 25\%}$	(S)	Men Women	1.79 ± 0.38 liter · sec^{-1} 1.96 ± 0.27 liter · sec^{-1}	109.0 ± 22.9	111.1 ± 26.9	109.3
	(E)	Men Women	108 ± 30% 98 ± 12%	101.6 ± 10.7	104.8 ± 20.1	103.0
FVC	(S)	Men Women	5.08 ± 1.14 liter 3.20 ± 0.60 liter	6 + 3 Cigarette experiment (n = 8)		
	(E)	Men Women	100 ± 9% 104 ± 8%			
$FEV_{1.0}$	(S)	Men Women	4.21 ± 0.46 liter 2.91 ± 0.36 liter			

	(I:)	Men Women	108 ± 36% 99 ± 12%	104.6 ± 10.7	106.4 ± 20.1	104.0

6 + 3 Cigarette experiment (n = 8)

FVC	(S)	Men Women	5.08 ± 1.15 liter 2.70 ± 0.60 liter	101.9 ± 4.4	100.9 ± 4.9	101.0
	(I:)	Men Women	110 ± 22% 108 ± 4%	96.0 ± 8.3	99.4 ± 8.1	97.8
$FEV_{1.0}$	(S)	Men Women	4.21 ± 0.96 liter 2.56 ± 0.56 liter	99.1 ± 9.4	101.1 ± 7.7	99.2
	(I:)	Men Women	99 ± 7% 98 ± 9%	99.4 ± 9.9	99.9 ± 7.0	100.6
$\dot{V}_{max\ 50}$	(S)	Men Women	3.66 ± 1.21 liter · sec $^{-1}$ 4.13 ± 0.92 liter · sec $^{-1}$	102.6 ± 8.9	102.9 ± 10.9	100.5
	(I:)	Men Women	109 ± 48% 95 ± 21%	106.1 ± 18.1	102.7 ± 15.9	103.9
$\dot{V}_{max\ 25}$	(S)	Men Women	1.48 ± 0.57 liter · sec $^{-1}$ 2.25 ± 0.44 liter · sec $^{-1}$	104.9 ± 16.9	112.1 ± 19.6	108.6
	(I:)	Men Women	124 ± 51% 86 ± 16%	102.9 ± 20.4	105.1 ± 27.1	99.9

[a] All data mean ± SD of values expressed as percentage of corresponding preexposure results. Findings pooled for men and women at each dose.
[b] Average based on observations after 0, 5, 10, 30, 60, 90, and 120 min of observation. Detailed data, with SD, available on request.
[c] Complete data not available for remaining subjects.
[d] Change relative to sham $P < 0.025$.
[e] Change relative to sham $P < 0.05$.

$\dot{V}_{max\ sm}$ to decrease by 7% or less (Clarke *et al.*, 1970, Da Silva and Hamosh, 1973; Zuskin *et al.*, 1974; Hamosh and Da Silva, 1977). The limited change in $\dot{V}_{max\ sm}$ has been attributed to (i) a reversal of spasm by the deep inspiration that precedes a $\dot{V}_{max\ sm}$ measurement and (ii) the peripheral nature of the airway spasm induced by cigarette smoke (Hamosh and Da Silva, 1977). The body plethysmograph used by Nadel and Comroe (1961) might also be thought a measure of large rather than small airway spasm. However, their data were presented as specific conductances (conductance/total thoracic gas volume) and peripheral spasm could have influenced the results through a change of mean alveolar volume.

There are many problems in calculating the "cigarette equivalent" for a "passive" smoker (Pimm *et al.*, 1978). Nevertheless it is unlikely that our subjects inhaled more than the equivalent of half a cigarette during their 2 hr in the exposure chamber. Thus, the maximum anticipated change, from previous studies of active smokers, would be a 3–4% decrease of $\dot{V}_{max}$. Further, in view of the onset of an anxiety hyperventilation, a part of any pharmacological response could be reversed by sympathetic nerve discharge and/or secretion of catecholamines. The observed response thus reaches at least the expected level.

Implications for Air Quality Criteria

The present experiments provide only meager evidence of respiratory responses to passive cigarette smoke exposure, despite the combination of high smoke concentrations and intermittent exercise. We would thus conclude that the main basis for setting air quality criteria is not the passing of a threshold for respiratory tract disturbances, but rather:

(a) subjective tolerance (odor and eye irritation, Shephard *et al.*, 1978a,c),

(b) possible impairment of fine vision (Shephard *et al.*, 1978b),

(c) possible chronic health consequences of nitrosamine exposure (Brunemann, 1978),

(d) possible lowering of angina threshold in patients with ischemic heart disease (Aronow, 1978), and

(e) possible sensitivity of the airways to tobacco smoke in patients with hyperreactive bronchi (Pimm *et al.*, 1977).

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